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Foreign Animal Disease Report

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**Emergency
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Foreign Animal Disease Investigations. A total of 46 investigations of suspected
foreign animal disease were conducted by veterinarians from the U.S. Department of
Agriculture, Animal and Plant Health Inspection Service, Veterinary Services, during the
period January 1 through March 31, 1989. The investigations were to eliminate the
possibility that an exotic disease had been introduced into the United States. All investi-
gations were negative for exotic diseases. An official report of each investigation of a
suspected foreign animal disease is required to be reported by a qualified Foreign Animal
Disease Diagnostician. *(Dr. John L. Williams, APHIS, USDA, Hyattsville, Maryland 20782,
(301) 436-8073)*

Avian Salmonellosis. Salmonellosis in chickens, caused by Salmonella enteritidis (SE)
phage type 4, has not been detected in the United States (see Foreign Animal Disease
Report 16-4:2-3). Other phage types of SE (types 7, 8, 19b, 13a, 14a, 14b, 23, and 28)
have been identified. Phage types 8 and 13a are the predominant types associated with
poultry in the United States. These types have a lower level of virulence in chickens than
type 4. Poultry losses and human health effects associated with SE phage type 4 in many
European nations underscore the need for import restrictions, rapid detection, and reliable
diagnostic capability to prevent the introduction of this agent into the United States.

A microbiologist from the National Veterinary Services Laboratories (NVSL), recently
trained in phage typing, has begun testing specimens collected in the United States.
Specimens collected previously were tested in the United Kingdom and Canada.

NVSL provides State and territory diagnostic laboratories with serologic reagents. Tube
test antigen antigen for *S. enteritidis* is available in addition to the pullorum microtiter test
antigen which is also used in serology for S. enteritidis. NVSL is in the process of prepar-
ing control antisera for *S. enteritidis* at both low and high titer. Some laboratories use
whole blood in the SE plate test. Serum is considered to give more reliable results.
Serum is considered the specimen of choice for the SE plate test.

In the United States, an interim regulation has been published in Title 9, Code of Federal
Regulations, Part 94, to restrict the importation of table eggs from all countries where *S.
enteritidis* phage type 4 is considered to exist. Canada is the only country outside the
United States where poultry is known to be free of *S. enteritidis* phage type 4, based upon
phage type surveillance. *(Dr. Sean F. Altekruse, APHIS, USDA, Hyattsville, Maryland
20782, (301) 436-8091)*

Training. A seminar for Foreign Animal Disease Diagnosticians (FADD) in the South-eastern United States is scheduled for June 20-22, 1989, in Athens, Georgia. This may be the only FADD seminar which will be held during the current fiscal year.

A seminar on wildlife diseases for Foreign Animal Disease Diagnosticians is scheduled for July 31-August 4, 1989, in Athens, Georgia. *(Dr. John L. Williams, APHIS, USDA, Hyattsville, Maryland 20782, (301) 436-8073)*

Foreign Animal Disease Update

In South America, during October, November, and December 1988, Brazil reported to the Office International des Epizooties (OIE) 176 outbreaks of foot-and-mouth disease (FMD) of types O and A; Argentina reported 159 outbreaks of types O, A, and C; Colombia reported 136 outbreaks of types O and A; Uruguay reported 4 outbreaks of FMD types O and C; and Ecuador reported 4 outbreaks of FMD type A.

In Europe, during November 1988, Italy reported 60 cases of FMD type C. The first cases were detected in swine on November 6, in Correggio. The following week, FMD was diagnosed in cattle, and 80 animals were destroyed. A secondary outbreak was confirmed on November 27, in Carpi, Italy, approximately 4 km from Correggio. The outbreak in Carpi affected only cattle and resulted in the destruction of 22 animals. On March 9, 1989, Italy confirmed yet another outbreak of FMD type C, but this time in Brescia. Brescia lies approximately 90 km to the north of Correggio and Carpi. All 74 bovines on the ranch were destroyed. Turkey reported 48 cases of FMD types O and A.

In Africa, Kenya reported 20 outbreaks of FMD types O, A, C, and SAT 2; South Africa reported 4 outbreaks of SAT 2; CHAD, Niger, and Benin reported the presence of the disease, but not the types; and Uganda reported outbreaks of FMD types O, A, C, SAT 1, and SAT 2. Egypt reported two outbreaks of FMD in November. Four hundred and sixty-one buffalo calves showed FMD lesions. Twelve thousand animals were vaccinated.

In the area that encompasses Asia, the Far East, and Oceania, Thailand reported 13 outbreaks of untyped FMD;Hong Kong, Oman, Kuwait, and Saudi Arabia reported FMD type O; Iran reported types O and A; and Pakistan reported outbreaks of FMD types O, A, and Asia 1.

The World Reference Laboratory for FMD, Institute for Animal Health, Pirbright Laboratory, Great Britain, reported the following diagnostic results for October, November, and December 1988:

Type O - Nepal, Hong Kong, Oman, Saudi Arabia, and Kuwait
Type C - Italy
Asia 1 - Nepal and Kampuchea

Vesicular stomatitis (VS), types New Jersey (NJ) and Indiana (IND), was reported from Central and South America. The following VS outbreaks were reported for the indicated country **:

	NJ	IND		IND	NJ
Mexico	1	1	Guatemala	50	10
Honduras	13	1	El Salvador	5	3
Nicaragua	11	0	Costa Rica	10	0
Panama	0	1	Colombia	42	36
Ecuador	2	0	Venezuela	1	0

** Data from Mexico, Central America, and Panama were reported to the Pan American FMD Center. Data from South America were reported to the OIE.

Swine vesicular disease (SVD) was reported from Hong Kong and Italy in December 1988. A total of 300 Italian swine were affected in the Venezia region. Italy had not reported SVD since 1984.

From October through December 1988, **rinderpest** (RP) was reported only on the continent of Africa: in Gabon, Ghana, Kenya, Uganda, Sudan, and Burkina Faso. Kenya reported 44,000 cases of RP in wildlife species.

Peste des petits ruminants (pest of small ruminants) was reported from Benin, Ghana, and Mauritania in Africa, and Oman, on the Arabian Peninsula.

Eight countries in Africa continued to report outbreaks of **contagious bovine** pleuropneumonia (CBPP): Benin, Ghana, Gabon, Kenya, Uganda, Namibia, Guinea, and Sudan. Kuwait also reported cases of CBPP.

Lumpy skin disease (LSD) was reported from eight African nations: the Congo, Zimbabwe, Mauritania, Niger, Kenya, Uganda, Mali, and Madagascar.

Cases of **Rift Valley fever** (RVF) were reported only from the African nation of Niger.

Outbreaks of **sheep and goat pox** (SGP) were reported from seven African countries: Tunisia, Morocco, Algeria, Mauritania, Niger, Kenya, and Mali. Pakistan, Kuwait, and Oman also reported cases. Turkey reported 134 new outbreaks of SGP involving 704,186 animals.

Cases of **African horse sickness** (AHS) continued to be reported from Spain during October, November, and December 1988, and January 1989. The last death of a horse due to AHS in Spain reportedly was on January 30, 1989. Location and extent of these cases were reported in the Spring 1989 issue of the Foreign Animal Disease Report *(see Foreign Animal Disease Report 17-1:2-6)*.

In Africa, Zaire and Uganda reported outbreaks of **African swine fever** (ASF). In Europe, Portugal reported 543 new outbreaks and sacrificed 19,460 swine; Spain had 249 new outbreaks and sacrificed 38,637 animals; and Italy reported 2 outbreaks involving 37 new cases.

Fifteen countries reported outbreaks of **hog cholera** (HC): in the Americas, Argentina, Ecuador, Paraguay, Uruguay, Chile, Colombia, and Brazil; in Mexico, 19 outbreaks with

274 new cases reported during the quarter; in Africa, Mauritius and Madagascar; in the area of Asia, the Far East, and Oceania, Malaysia and the Philippines; and in Europe, Yugoslavia, Italy, and France. France reported 14 outbreaks in 6 different departments (States) where 5,359 swine were destroyed.

Teschen disease (TD) was reported from Madagascar and the Soviet Union.

Fowl plague (FP) was reported in Africa from Benin, Niger, and Mauritania.

Thirty-one countries worldwide reported outbreaks of **Newcastle disease** (ND) (not typed and presumed to be velogenic). ND was reported from Africa in Egypt, Algeria, Tunisia, Ghana, Gabon, Congo, Guinea, Berkina Faso, Niger, Zaire, Kenya, Uganda, Botswana, South Africa, and Madagascar. In the Americas, this disease was reported from Mexico, Panama, Ecuador, Colombia, and Brazil. ND was reported in Europe from Italy, Turkey, and Yugoslavia. In the area of Asia, the Far East, and Oceania, untyped ND was reported from the Philippines, Taiwan, Hong Kong, Republic of Korea, Malaysia, Pakistan, Kuwait, and Iraq.

Velogenic viscerotropic Newcastle disease (VVND) was reported from Guinea, Gabon, Cyprus, the Soviet Union, Malaysia, Pakistan, and Indonesia. A total of 531,517 new cases of VVND were reported during the outbreaks in Indonesia. Cyprus reported that VVND was introduced through imported captive birds from the Netherlands in December, with subsequent spread to the Nicosia Municipal Garden's captive birds in January 1989.

Information reported for the world animal disease situation during October, November, and December may reflect previously unreported data from outbreaks which occurred in previous months. *(Dr. E. R. Hoffman, APHIS, USDA, Hyattsville, Maryland 20782, (301) 436-8892)*

Heartwater

Background. Heartwater is a disease of ruminants causedby *Cowdria ruminantium*, a rickettsial parasite transmitted by several tick species of the genus *Amblyomma*. The main vectors are *A. variegatum*, the tropical bont tick, and *A. hebraeum*, the southern African bont tick. These ticks and the disease were originally confined to Africa, but in the last two centuries there has been some spread caused by the movement of livestock. The tropical bont tick is now well established on Madagascar and various small islands in the Indian and Atlantic Oceans and the Caribbean. Heartwater has been confirmed on Madagascar and several of the small islands, including three in the Caribbean (Antigua, Guadeloupe, and Marie Galante).

Normally, heartwater is not a problem in indigenous livestock which are born in endemic areas and acquire immunity through natural exposure when young. The disease sometimes appears in endemic areas when vector numbers are too low to ensure that all young animals become infected. Numbers of *Amblyomma* ticks may be low because of unsuitable ecological conditions or the use of acaricides for tick control. Unacceptably high losses from heartwater also occur when highly susceptible breeds, such as Angora goats, are raised in endemic areas.

There is an age-related resistance which lasts for about a month after birth. Heavy mortality, often exceeding 50 percent, occurs in cattle, sheep, and goats if older susceptible animals are exposed to the disease. Such exposure may be due to stock being moved from localized heartwater-free areas to endemic areas, exotic animals being brought into endemic areas, and infected vectors spreading to new areas.

Control. Specific control measures against heartwater are required only to protect stock brought into endemic areas, stock in areas that are ecologically marginal for Amblyomma ticks, stock that is subjected to intensive acaricide treatment for the control of other tick-borne diseases such as East Coast fever (caused by *Theileria parva*), and susceptible breeds that are kept in endemic areas. Measures such as serological screening and tick control should also be taken to prevent the spread of heartwater and its vectors to new areas through the translocation of domestic or wild hosts.

Effective control of heartwater is frequently difficult to achieve. Vector eradication by acaricide treatment of domestic hosts is impossible if untreated wild hosts are present, as is the case through much of Africa. The only method of immunization available at present is infection and treatment. The infective material, usually in the form of either whole blood from an infected animal or tick stabilate, produces severe reactions. Animals must be individually monitored after infection so that tetracycline treatment can be administered at the start of the febrile reaction. Block treatment of herds or flocks cannot be carried out, as the incubation period is extremely variable. Treatment prior to the febrile reaction will prevent the development of immunity, and treatment too late in the reaction can result in death. Losses in cattle, sheep, and goats of as much as 30 percent of infected groups have occurred in Zimbabwe and South Africa where heartwater immunization has not been carried out under strict veterinary supervision. Failure to control heartwater has also been the result of the misunderstanding of several important aspects of the epidemiology of the disease.

Epidemiology. Studies on the epidemiology of heartwater have been hampered until very recently by the absence of serological or other tests to determine the presence of *C. ruminantium* in hosts or vectors. The cause of the problem has been the lack of an adequate source of the organism. Fortunately, this has now been resolved by the *in vitro* culture of *C. ruminantium* in bovine endothelial cells, first described by scientists at the Veterinary Research Institute, Onderstepoort, South Africa, in 1985. Several different isolates have since been grown in culture in South Africa, Zimbabwe, and Kenya. Sero-logical tests have been developed using purified antigens. A new phase in research on the epidemiology of heartwater is beginning.

For many years, the carrier state was considered not to exist in heartwater disease. Hosts were generally accepted to be infective during the febrile reaction and for a short period thereafter. However, scientists of the U.S. Agency for International Development (USAID), University of Florida/Zimbabwe Heartwater Research Project in Harare, recently showed that domestic and wild ruminants can remain long-term carriers of *C. ruminan-tium*. Sheep and cattle were infective for *A. hebraeum* nymphs at 8 months post-innocula-tion (p.i.), and African buffalo (*Syncerus caffer*) at 5 months p.i. Testing for longer periods has not been carried out. Previous studies, indicating the absence of the carrier state, involved attempts to transmit the agent using either nymphs fed as larvae on recovered animals or blood from recovered animals. It is possible that nymphs pick up low-level infections, whereas larvae do not. Workers at the International Laboratory for Research on Animal Diseases (ILRAD) in Nairobi, Kenya, have shown that *Cowdria* organisms multiply in the gut epithelial cells of nymphs of *A. variegatum*. This has a concentrating effect and may not occur in larvae.

The role and relative importance of wildlife in the epidemiology of heartwater requires clarification. Some species have been shown to be heartwater-susceptible, but others have not; a number (including some nonruminants) have been found to harbor C. rumi-nantium for short periods. For many years, wildlife was suspected to be an important reservoir of infection; this was recently supported by results of laboratory work with buffalo

in Zimbabwe. Interestingly, the African buffalo is one of the species in which there is no clinical reaction to heartwater.

Another long-held belief was that heartwater infection rates in vector populations were low; normally less than 5 percent. This has been disproved by an investigation recently undertaken by the USAID-funded project in Zimbabwe, involving unfed nymphs and adults of *A. hebraeum*. Small pools (about 5) of field-collected ticks were allowed to feed on known susceptible sheep, some of which became infected. The proportions of infected ticks were then estimated statistically. The results from ticks collected at two localities were nymphs 1-10 percent infected, adult males 10-20 percent infected, and adult females 20-40 percent infected. These were obviously minimum estimates, as the tests could have false negatives, but not false positives. It will clearly be of value to carry out further studies on infection rates when it is possible to determine the presence of *C. ruminantium* in ticks by direct methods such as DNA probes.

Host location and selection by the tick vectors are also important in the epidemiology of heartwater. These have been studied in Zimbabwe. Unfed nymphs and adults of *A. hebraeum* are activated by CO_2, but only move toward and attach readily to hosts if there are already fed males on them. The response of the unfed ticks is to a phero-mone that is emitted by the males after a period of feeding. It allows unfed ticks to discriminate between suitable hosts, on which males have fed successfully, and potentially unsuitable hosts, on which there are no fed males. Unsuitable hosts are those on which the ticks are unlikely to survive due to resistance or grooming and other factors, such as regular treatment with acaricides. The response to the phero-mone thus minimizes the effectiveness of acaricides in reducing tick populations, as it causes unfed ticks to attach to untreated alternate hosts (e.g., wild antelope) in preference to treated domestic animals.

Prospects for control in Africa. Under African conditions, heartwater is a disease that lends itself to management rather than eradication. This is because there are large reservoirs of infection in both the host and vector populations, and the mecha-nism of host selection by vectors makes their eradication by acaricides impossible if alternate wild hosts are present. In addition, intensive control of the heartwater vectors can be counterproductive because it will reduce the chances of young animals becoming naturally immunized. *Amblyomma* ticks, although large and damaging, seldom occur in high enough numbers on indigenous livestock to justify the costs of frequent acaricide treatment; occasional treatment of heavy infestations is all that is required. It is only when tick-susceptible exotic animals, such as *Bos taurus* cattle, are introduced that the need arises to control these ticks.

The most useful management tool would be a safe and effective vaccine which could be used to immunize susceptible stock being moved into endemic areas, young stock born in marginal or unstable situations, or stock that is threatened by the spread of the disease. The principal aim of the USAID-funded project in Zimbabwe is to develop such a vaccine. Research that could also lead to the development of an improved vaccine is being carried out by the Kenya Government in collaboration with Washing-ton State University and the Veterinary Research Institute, Onderstepoort.

Heartwater in the Western Hemisphere. Although the tropical bont tick was intro-duced to the Caribbean from Africa in about 1830, with Senegalese cattle shipped to the French Antilles, it was only recently positively diagnosed there. It was first estab-lished on Guadeloupe and Antigua, and later spread to other islands, reaching Puerto Rico, St. Lucia, and Barbados.

The presence of heartwater and *A. variegatum* in the Caribbean poses a real threat to the American mainland. This is because the tick is spreading, and large areas of North, Central, and South America have been shown by a computerized climate-matching model (CLIMEX) to be suitable for its survival. Factors that are thought to be causing the spread are increased transportation of livestock between islands and increased abundance of the cattle egret (*Bubulcus ibis*), an introduced bird from Africa, which is frequently infested with the immature stages of *A. variegatum*.

Heartwater could be introduced to the mainland by migratory birds bringing infected *A. variegatum* nymphs or with ruminants carrying *C. ruminantium*, and possibly ticks. Carrier animals alone may be sufficient to allow the establishment of the disease as it has been shown in laboratory studies that two widely distributed *Amblyomma* species, the cayenne tick (*A. cajennense*) and the Gulf Coast tick (*A. maculatum*), can serve as vectors. With vectors already present on the mainland, there is also the danger that infection could become established from wild ruminants or other carriers imported from Africa. Ideally, potential hosts from endemic areas should not be imported until it has been confirmed that they are not carriers. Improved diagnostic tests should make this possible in the forsee-able future.

Since heartwater is an introduced disease in the Western Hemisphere and still restricted to limited island foci, it would be desirable to eradicate it rather than risk its spread. This could be done, as is already appreciated by the veterinary authorities in the region, by the use of acaricides to eradicate *A. variegatum*. Eradication of the tick should be possible, because there are very few alternate hosts on the islands. Consideration could also be given to the use of the male-produced pheromone to increase the attractiveness of treated animals to unfed nymphs and adults. *(R.A.I. Norval, International Laboratory for Research on Animal Diseases, P.O. Box 30709, Nairobi, Kenya, formerly Veterinary Research Laboratory, P.O. Box 8101, Causeway, Zimbabwe, Africa)*

Necrotic Hepatitis of Rabbits

In December 1988, two outbreaks of a rapidly spreading fatal disease of domestic rabbits were reported in Mexico City, Mexico (see Foreign Animal Disease Report 17-1:8-9). One was in the vicinity of a supermarket which had received a shipment of rabbit meat from the People's Republic of China on November 19, 1988. The disease spread rapidly, extending as far as 270 miles from the initial outbreaks, within 100 miles of the Texas border. Ten States in Mexico were affected and 595 premises determined to have the disease, with 18,000 deaths reported by the end of March 1989. Eradication programs were undertaken, including slaughter and disinfection. All rabbits from positive foci were eliminated in 5 of the 10 affected States. By April 30, 1989, more than 33,838 rabbits reportedly had been sacrificed, and 40,620 rabbits had died from necrotic hepatitis in Mexico.

The signs reported in most of the outbreaks in Europe and Asia include an acute disease with high fever, respiratory distress, and bloody nasal discharge, with death following a few hours after the onset of clinical signs. Many rabbits were found dead with no previous signs of illness. Morbidity was estimated at 30-80 percent, with mortality reaching 80-90 percent. Although the relatedness of outbreaks in different countries is not confirmed, and the causative agents have not yet been fully identified, the clinical signs, epizootiology, and pathologic findings suggest a common etiological agent.

A small virus, reported to be 30-35 nm, was identified in rabbit tissues from various outbreaks. The disease has occurred in the People's Republic of China since 1984, and has been attributed to a picornavirus or calicivirus and, more recently, to an unclassified virus called rabbit hemorrhagic disease virus (RHDV). In Korea, a picornavirus was

suggested as the cause of a similar disease. In Germany, a parvovirus was suggested as the cause of X disease of the rabbit. A similar disease in Switzerland was called necrotic hepatitis of rabbits.

Another disease syndrome reported from Sweden having similar clinical signs and pathological lesions was called European brown hare syndrome. Initially, a toxic etiology was suspected for this syndrome, and no virus has yet been demonstrated.

Diagnostic activities. On February 10, 1989, fresh and formalin-fixed tissue samples from field and inoculated rabbits with necrotic hepatitis were sent by Dr. John Mason, Dr. Farouk Hamdy, and Dr. Juan Lubroth of the Mexico-United States Commission for Prevention of Foot-and-Mouth Disease and Other Exotic Diseases (CPA), United States Department of Agriculture (USDA), in Mexico City, to the Animal and Plant Health Inspection Service (APHIS) Foreign Animal Disease Diagnostic Laboratory (FADDL) at Plum Island, New York. The disease was reproduced in New Zealand White rabbits by intranasal and intramuscular routes of inoculation, using a bacteria-free filtrate of a liver homogenate from the submitted field case. Within 24 to 48 hours, the rabbits were febrile and all died within the next 24 hours with terminal signs of respiratory distress and a bloody nasal discharge.

Pathology. Necropsy findings in field cases and inoculated rabbits were relatively subtle. The livers were pale tan to slightly mottled and friable, with a lobular pattern of congestion. The spleens were engorged and approximately two to three times normal thickness. Pale foci were noted in the myocardium. In rabbits that died, some lungs were edematous and congested, with blood-tinged fluid in the airways. Petechial hemorrhages were seen on some lung surfaces and one mesenteric lymph node.

Histopathologic lesions were more remarkable. There was moderate multifocal-to-massive necrosis of the liver, which accounted for the mottled pale tan color seen on gross examination. The splenic red pulp was congested with abundant fibrin in the sinusoids and numerous scattered necrotic cells. Necrosis in the white pulp was variable, involving just the marginal zone or, in field cases, the entire lymphoid tissue. There were multiple focal areas of necrosis in the myocardium. The lungs were severely congested and edematous, perhaps as a result of myocardial damage. In the tissue submitted from Mexico, there was severe crypt necrosis in the small intestine; however, this was less evident in inoculated rabbits. The latter lesion with focal myocardial necrosis is similar to that seen in the early cases of canine parvovirus in puppies. This finding, along with the demonstration of a 28 nm viral particle with little surface morphology in liver homogenate, and the ability of tissue homogenate to hemagglutinate human red blood cells, suggested that this may be a parvovirus.

Virus. Further definitive evidence was found with electron microscopy. Intranuclear dense granular to globular inclusion bodies with closely associated linear arrays of 25-30 nm viral particles were found in the nucleus of degenerate hepatocytes and splenic lymphocytes, which is typical of the small single-stranded DNA parvoviruses.

Serologic relatedness of this suspected parvovirus to other known parvoviruses was tested using a panel of monoclonal and polyclonal antisera to various parvoviruses of rodents, pigs, dogs, cats, and mink supplied by Dr. Abigail Smith, Yale University; Dr. William Mengeling, National Animal Disease Center; Dr. Richard van Deusen, formerly of the National Veterinary Services Laboratories; and Dr. Leland Carmichael, Cornell University. Using avidin-biotin alkaline phosphatase immunostaining on infected rabbit spleen sections and hemagglutination inhibition with human type-O red cells, the virus

was characterized as an unclassified virus with some degree of antigenic relatedness to a minute virus of mice and porcine parvovirus.

Attempts to isolate the virus in vitro using a wide variety of cell lines of rabbits, other laboratory animals, and domestic animals, as well as primary rabbit cell cultures and organ cultures, have been unsuccessful. Suckling mice and embryonating eggs are also resistant to infection.

Diagnostic tests. Since the mortality rate of this rabbit disease is so high, a diagnosis of a disease outbreak will most likely be made on the basis of gross pathology, histopathology and hemaglutination of human red blood cells with clarified liver or spleen homogenates. A diagnosis may be confirmed with direct immunostaining of spleen sections. Due to the difficulty encountered in propagating this virus, a rapid serologic test is not yet available.

Prevention and control. No commercial vaccine is yet available for this new disease. As a prophylactic measure, autologous killed vaccines have been shown to be effective under experimental conditions in the People's Republic of China, but were not effective in the face of an outbreak. Various vaccine trials are presently underway at FADDL.

The only effective control measure at present is prevention. Rabbit raisers should be made aware of the potential risk of introducing this disease into the United States. New stock from outside the United States should be quarantined for at least 1 week before introduction into a rabbitry. This is simply a good management practice at any time.

Once introduced, the only means of control, at present, is depopulation and disinfection. This virus, like other parvoviruses, is extremely stable in the environment and is resistant to mild disinfectants and pH change. Cleaning must be thorough, with careful removal and disposal of all fecal material and bedding. The recommended disinfectants are 1-Stroke Environ, and Vanadine. Repopulation of previously infected premises should follow a waiting period of 2 months and would best be limited at first to a few sentinel rabbits before complete repopulation is considered.

Human exposure. No human disease has been attributed to this new virus. A number of field workers and laboratory workers in various countries have been exposed to the virus, with no infections reported. Animal inoculation experiments suggest that this virus may only infect rabbits.

Wild rabbit populations. Wild rabbits and hares of various species abound throughout the United States and Mexico. Should wild rabbits be susceptible to this new disease, an outbreak in domestic rabbits could be amplified many-fold, and the disease could be rapidly spread by wild populations. The susceptibility of wild cottontail rabbits is presently under study at FADDL. Susceptibility studies with other wild species may follow. So far, the virus of necrotic hepatitis of rabbits has not been found in wild host species.

Similar diseases. Disease syndromes in rabbits with similar losses and pathologic lesions have been reported in various European and Asian countries. A variety of names have been used in the literature to date. These include: viral hemorrhagic disease of rabbits; necrotic hepatitis of rabbits; X disease of rabbits; hemorrhagic pneumonia in rabbits; and infectious hemorrhagic disease of rabbits. The degree of hemorrhage and pulmonary involvement seems to vary among the different reports. Perhaps secondary bacterial or viral infections are responsible for these differences. In our experience with the Mexican isolate, hemorrhage has not been prominent and pulmonary congestion is

more likely a secondary lesion resulting from myocardial necrosis. For these reasons, we have chosen to use the more descriptive name, necrotic hepatitis of rabbits, for this parvoviral disease. (Dr. D. Gregg, Dr. T. Wilson, and Dr. C. House, USDA, APHIS, S&T, NVSL, FADDL, Box 848, Greenport, NY 11944)

Focus on Melioidosis

Melioidosis is an infectious, glanders-like disease of man and animals, endemic to certain areas of the tropics and subtropics. The clinical picture ranges from a mild, chronic disease with localized lesions to a rapidly fatal septicemic form. Latent infections are common, with clinical signs developing months or years after the initial exposure.

The causative agent of melioidosis is Pseudomonas pseudomallei, formerly known as *Bacillus pseudomallei*, *Loefflerella whitmori*, *Pfeifferella whitmori*, and *Malleomyces pseudomallei*.

P. pseudomallei is a small, gram-negative, bipolar staining, pleomorphic bacillus. The organism grows well on routine laboratory media. Colonies may vary from smooth to rough and wrinkled in texture, and from cream to bright orange in color. Motility is easily demonstrated in wet mount preparations. Like other aerobic pseudomonads, *P. pseudomallei* is oxidase-positive and produces acid from the oxidation of glucose and other carbohydrates. Strains also reduce nitrate with the production of gas, liquify gelatin, and peptonize litmus milk. Soluble pigments and indole are not produced.

P. pseudomallei is susceptible to common disinfectants, such as iodine, sodium hypochlorite, and benzalkonium chloride.

Human Melioidosis

The first description of melioidosis is credited to Whitmore and Krisnaswami, who encountered it in vagrants and morphine addicts in Burma in 1912. Whitmore proposed the name *Bacillus pseudomallei* for the etiological agent because of its morphological and pathological similarities to the glanders agent.

The disease has received some attention in recent years because of its appearance in some veterans of the Vietnam War.

The incubation period in humans is unknown. Information obtained from patients whose time of exposure was known suggests it may be from 4 to 5 days. However, some individuals may harbor the organisms for months or years before clinical disease appears.

The clinical symptoms of melioidosis in man are often vague. The disease may be confused initially with pneumonia, cholera, typhoid, plague, malaria, tuberculosis, glanders, or other diseases. For convenience, the disease may be classified as either acute, subacute, chronic, or inapparent.

The relatively rare acute form of melioidosis is characterized by sudden onset of symptoms, primarily fever, chills, and prostration. Severe gastroenteritis, pneumonitis, and septicemia develop, followed by death in 2 to 4 days.

More often, the disease is subacute, with a course of a few days to several months. The usual primary sign of infection is either a skin lesion at the portal of entry, with subsequent lymphangitis, regional lymphadenitis, and septicemia, or pneumonitis followed by septicemia and generalized organ involvement. Symptoms depend on the organ system involved and may include any combination of the following: pneumonia,

myocarditis, hepatitis, meningitis, or osteomyelitis. (*Brundage, W. C., et al. 1968. Am. J. Trop. Med. Hyg. 17(2):183-191*)

The chronic form of melioidosis may develop in patients who survive septicemic infection, or may arise indolently. Clinically, this form resembles tuberculosis or systemic mycosis. Symptoms include fever, productive cough, weight loss, and anemia. Radiographs show upper-lobe nodular infiltrates, often with cavitation. Localized abscesses may also develop subcutaneously and in bones, joints, or viscera. Chronic infections may flare into acute fulminant septicemia months or even years after the initial infection. Conditions which lower host resistance, such as trauma, surgery, or debilitating illness, are often associated with reactivation. (*Prevatt, A. L., and J. S. Hunt, 1957. Am. J. Med. 23:810-823; Morrison, R. E., et al. 1988. Am. J. Med. 84:965-967*)

Inapparent infections, expressed only by a serologic response, are common in the native populations of endemic areas. Significant hemagglutination titers have been found in as many as 29 percent of healthy indigenous persons from selected population groups in Malaysia and Thailand. Titers were more common in males from rural areas, probably as a result of occupational contact with the soil. (*Howe, C., et al. 1971. J. Inf. Dis. 124 (6):825-833*)

Nonhuman Melioidosis

Naturally acquired melioidosis has been reported in numerous species of domestic and wild animals, including dogs, cats, cattle, pigs, sheep, goats, horses, deer, rats, and nonhuman primates. The disease has been produced experimentally in guinea pigs, mice, hamsters, and rabbits. Melioidosis is seldom reported in avian species. In most instances, the disease is sporadic, but outbreaks have been reported in sheep, goats, and pigs in some endemic areas.

Although acute melioidosis is often reported in animals, serologic and slaughter inspection surveys indicate that chronic and inapparent infections are the most common forms of the disease. Acute disease, which results in death of the animal, is usually the result of activation of chronic infection. Stress resulting from transportation, pregnancy, lactation, or debilitating disease can precipitate acute episodes of melioidosis. Acute disease without underlying chronic infection is usually seen only in young animals.

Clinical signs are nonspecific. Acute melioidosis usually manifests as a bronchopneumonia with subsequent septicemia. Principal clinical signs are depression, fever, cough, and nasal discharge. Death occurs within a few days. Cattle with acute melioidosis have developed endometritis and placentitis.

Chronically infected goats show progressive emaciation, lameness, and CNS signs. The organism often becomes established in the udder and causes mastitis. Affected sheep show lameness and respiratory signs.

In market swine, the disease is usually asymptomatic, and is not diagnosed until slaughter. The disease can cause significant economic losses to swine producers due to the condemnation of affected carcasses. There are a few reports of *P. pseudomallei* orchitis in boars. Melioidosis is uncommon in breeder swine, possibly due to an acquired immunity.

In horses, the clinical signs most often involve the respiratory tract or the lymphatic system. Colic and acute meningoencephalitis have also been associated with melioidosis in horses. (*Thomas, A. D. 1981. Aust. Vet. J. 57:146-148*)

Melioidosis was observed in military dogs during the Vietnam War. Affected animals exhibited fever, muscle pain, dermal abscesses, and epididymitis. (*Moe, J. B., et al. 1972. Am. J. Trop. Med. Hyg. 21(3):351-355*)

Melioidosis

Pathology

Melioidosis lesions include pyemia, with multiple abscesses in the lungs, lymph nodes, spleen, and less often, other viscera, skin, bone, and joints. The typical lesion begins as a small granulomatous nodule with caseous center. Some nodules become abscesses and contain creamy or caseous yellow-green pus. Lung lesions tend to coalesce and may cavitate, resembling pulmonary tuberculosis. Abscesses and ulcers in the nasal mucosa similar to those of equine glanders have been observed in sheep and goats. *P. pseudomallei* appears to have some predilection for testicular tissue. Guinea pigs inoculated intraperitoneally develop bilateral orchitis (Strauss reaction). Granulomatous orchitis is also commonly present in affected goats and boars.

Diagnosis

Because the clinical signs of melioidosis may be vague, diagnosis depends upon the identification of the etiologic agent, either by direct culture or by hamster or guinea pig inoculation. P. pseudomallei may be isolated from abscesses, blood, cerebral spinal fluid, sputum, synovial fluid, and urine. Cultural identification may be confirmed with fluorescein-conjugated antiserum. This serum will cross-react with P. mallei. The organisms may be differentiated by *P. mallei's* lack of motility. (*Howe, C., et al. 1971. J. Inf. Dis. 24(6):825-833*) Complement-fixation and indirect hemagglutination tests may provide additional confirmation of infection, but do not distinguish between melioidosis and glanders (*Alexander, A. D., et al. 1970. Applied Microbiol. 20(5):825-833*)

Geographic Distribution

Melioidosis is a disease of the tropics and subtropics. Most cases have been reported from Malaysia, Vietnam, and Australia. Sporadic cases have also been reported from other tropical areas, including the Philippines, Sri Lanka, India, Madagascar, Central and South America, and the Caribbean. (*Redfearn, M. S., et al. 1966. J. Gen. Microbiol. 43:293-313*) Endemic melioidosis was once considered limited to the area which extends from 20° N to 20° S of the Equator. Recently, endemic foci have been established in France, Iran, and southern Australia through the introduction of carrier animals. Melioidosis has not been reported in domestic animals in the United States. Human infections in the United States have, with few exceptions, been the result of exposure during military service in Vietnam or travel to an endemic area.

Transmission

The natural reservoirs of *P. pseudomallei* are soil and water. The organism usually enters the host through skin abrasions, but may also be ingested or inhaled. Cases of pulmonary melioidosis observed in Vietnam in helicopter crewmen were thought to have been initiated by the inhalation of organisms in dust raised by the helicopter rotors. (*Howe, C., et al. 1971. J. Inf. Dis. 124(6):598-606*) Laboratory workers have also acquired melioidosis by the inhalation of infectious aerosols. (*Green, R. N., and Tuffnell, P. G., 1968. Am. J. Med. 44:599-605*)

P. pseudomallei infections are common in pigs raised on contaminated soil, and outbreaks have occurred in swine raised under confinement with contaminated water supplies. Sheep and goats are commonly infected through wounds, such as those resulting from hoof paring and tail docking. (*Thomas, A. D., et al. 1981. Aust. Vet. J. 57:144-145*)

Other modes of infection are either rare or have only been observed experimentally. A case of venereal transmission was reported in man. (McCormick, J. B., et al. 1975. Ann. Int. Med. 83:512-513) Arthropod-borne infections, while not known to occur naturally, have been demonstrated experimentally in laboratory animals. (Laws, L., and Hall, W. T. K., 1964. Aust. Vet. J. 40:309-314)

P. pseudomallei has been isolated from kids born to experimentally infected goats, suggesting possible vertical transmission. (Thomas, A. D., et al. 1988. Aust. Vet. J. 65:43-47) It is possible that these modes of transmission are more common or that other unreported methods might exist. The exact mode of transmission is often difficult to determine because of the latent nature of this disease.

P. pseudomallei is usually highly resistant to drugs. Antibiotic therapy may be ineffective, particularly in cases of acute fulminant septicemia. Pulmonary and other localized infections have been treated successfully with antibiotics. The choice of antibiotic should be based on sensitivity tests. Tetracycline, chloramphenicol, novobiocin, kanamycin, sulfonamides, and trimethoprim-sulfamethoxazole have been shown to be effective against *P. pseudomallei* in vitro. There is considerable evidence from clinical reports that massive dosages for prolonged periods are required to eliminate the organism from all tissues and prevent relapse. Treatment may be complicated by toxic drug reactions or the emergence of bacterial resistance during therapy. (*Eickhoff, T. C., et al. 1969. Health Lab. Sci. 6:27-39*) Surgical drainage of abscesses or lung resection may be required in conjunction with antibiotic therapy.

Food animals are not usually treated for melioidosis because of the expense of long-term therapy and the public health significance of the disease. Dogs have been successfully treated with an extended course of tetracycline. (*Moe, J. B., et al. 1972. Am. J. Trop. Med. Hyg. 21(3):351-355*)

Melioidosis Treatment

Vaccines for melioidosis are not available. In endemic areas, prevention is aimed primarily at reducing host contact with contaminated soil and water. Ordinary sanitary precautions, such as prompt treatment of wounds, separating affected animals from healthy animals, and cleaning and disinfection of pens may reduce the incidence of infections. Efforts must also be directed at preventing the organism from becoming established outside its traditional habitat. There is nothing about the physiology of *P. pseudomallei* that would prevent it from thriving in soils of the United States. Animals originating in endemic areas, particularly sheep, goats, and nonhuman primates, should be subjected to serologic testing before importation.

Public Health Aspects

P. pseudomallei has been isolated from normal goat milk and from swine carcasses at slaughter. Although consumption of contaminated meat or milk has not been associated with human infections, contaminated food products may represent a public health risk. (Dr. L. K. Schlater, National Veterinary Services Laboratories, ST, APHIS, USDA, P. O. Box 844, Ames, Iowa 50010)

Editorial Committee	Editorial Committee membership has been changed. The current members are:
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